Shock: aetiology, pathophysiology and management

Daniela Blumlein and Ian Griffiths

ABSTRACT

The term 'shock' is used to describe a complex, life-threatening clinical condition that arises from acute circulatory failure. Shock is a pathological state that results when the circulation is unable to deliver sufficient oxygen and nutrients to the cells and tissues. The resulting hypoxia, tissue hypoperfusion and cellular dysfunction can lead to multi-organ failure; if this is not treated in a timely and appropriate manner, it can lead to death. This article gives an introduction to shock with an overview of the condition and its physiological impact on patients. Focusing on the aetiology and underlying causes, discussion will highlight the different types, stages and general pathophysiology of shock, as well as providing a guide to treatment options and nursing interventions.

Key words: Shock ■ Acute circulatory failure ■ Compensatory mechanisms ■ Hypovolaemia ■ Sympathetic response

Shock is best described as a severe, life-threatening form of acute circulatory failure characterised by inadequate tissue perfusion resulting in systemic hypoxia and cellular dysfunction. It is a pathological state caused by the circulation being unable to deliver sufficient oxygen and nutrients to the tissues and cells (Stratton, 2022).

Shock can result from traumatic injury or disease, creating a state of insufficient oxygenation and perfusion of vital organs throughout the body (Migliozzi, 2017). The condition affects up to one in three patients in critical care environments. Once diagnosed, its treatment relies on the rapid initiation of fluid resuscitation and often includes the use of vasoactive medications to improve cardiac output and tissue perfusion status (Scheeren et al, 2021).

Shock is a condition that requires nurses to make timely, evidence-based decisions for their patients. It is therefore vital that nurses can recognise shock as it happens and assess and understand the signs and symptoms of its various causes in order to initiate individualised treatments and therapies, in accordance with the professional standards required by the Nursing and Midwifery Council (2018) *Code* to improve patient outcomes and potentially save life.

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Aetiology of shock

Patients can experience shock for a number of reasons, including physical trauma, blood loss, dehydration or allergic reaction (Tait, 2022). Shock is used as an overarching term to describe a patient in a critical state of deterioration, so it is vital to first recognise and understand the type of shock being presented, which is typically categorised by causative factors (Migliozzi, 2017).

There are four main types of shock: hypovolaemic, cardiogenic, obstructive and distributive. Distributive shock is classified according to its three main individual causes, which relate to sepsis, a neurogenic disorder or anaphylaxis. Each type can be categorised by its individual cause and sub-type (*Table 1*).

While the aetiology of shock is varied and complex in nature, the fundamental pathophysiological process shared by each type means that, if left untreated, each individual root cause will lead to a reduced cardiac output and decreasing regional blood flow, tissue perfusion and overall oxygenation (Cecconi et al, 2014). Once this situation has developed, cellular hypoxia followed by acidosis and anaerobic metabolism will ensue.

Hypovolaemic shock

This is caused by a decrease in circulating blood volume. Considered to be the most common form of shock (Dutton and Elliot, 2021), hypovolaemic states are characterised by an inadequate intravascular volume caused by significant blood and/or fluid loss. Shock will occur when the circulating volume falls to a point at which the body's metabolic requirements cannot be met (Richards and Edwards, 2014).

Hypovolaemic states can occur owing to haemorrhage visible outside the body and, less discernibly, that which occurs within the body. The circulating volume can also be be altered by further causes of hypovolaemia, including plasma loss from extensive burns, fluid depletion because of dehydration, vomiting or diarrhoea, and internal fluid shifting such as occurs in peritonitis.

A significant decrease in circulatory volume leads to a lower volume of blood returning to the heart, decreasing cardiac output and reducing blood pressure (Migliozzi, 2017).

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Table 1. Main types of shock

Type of shock	Sub-type and causative factors
Hypovolaemic	External or internal fluid loss ■ Haemorrhagic (blood loss) ■ Non-haemorrhagic (plasma loss, dehydration, internal fluid shifting)
Cardiogenic	Impaired cardiac function ■ Cardiomyopathies (myocardial infarction, contusion, valve/septal defects) ■ Arrhythmias (electrocardiogram derangements)
Obstructive	Obstruction to circulation ■ Tamponade/mechanical ■ Pneumothorax/pulmonary ■ Emboli/vascular
Distributive	Maldistribution of blood flow/loss of vascular tone ■ Sepsis/systemic inflammatory response ■ Neurogenic/autonomic dysfunction ■ Anaphylaxis/hypersensitivity reaction

Source: adapted from Docherty and Hall (2002)

Cardiogenic shock

This is caused by impaired cardiac function. Even if fluid volumes are adequate, poor cardiac output can result in tissue hypoxia in situations where the heart fails to pump blood effectively throughout the systemic circulation.

This type of shock can occur because of cardiac arrhythmias and myorcardial impairment such as necrosis and heart valve dysfunction following myocardial ischaemia or infarction as a result of heart failure (Cecconi et al, 2014).

Obstructive shock

This is caused by obstruction to the circulating blood flow. Decreased cardiac output can have a physically obstructive cause that impedes blood flow and affects both the preload and afterload of the cardiac cycle. Such obstructions can be caused by numerous conditions, including cardiac tamponade, tension pneumothorax, pulmonary embolus, heart valve stenosis and certain anatomical defects that are more regularly observed in paediatric patients (Tait, 2022).

Distributive shock

This is caused by an altered distribution of circulating blood. It results from a number of conditions that cause blood vessels to lose their ability to maintain systemic vascular resistance and tone, which leads to a decrease in organ perfusion (Dutton and Elliot, 2021).

Distibutive shock has three main causes—septic shock, neurogenic shock and anaphylactic shock.

Septic shock, its principle cause, is a result of a widespread dysregulated response to an infection or insult, typically associated with the systemic inflammatory response syndrome.

Neurogenic shock is the loss of sympathetic nervous system activity (motor and sensory nerve impulses) because of brain or spinal cord injury, spinal anaesthesia and certain neuropathies including transverse myelitis and Guillain-Barré syndrome. While this condition creates a state of shock, it is vital that practitioners recognise that the usual stress response will not be observed (Dutton and Finch, 2018).

Anaphylactic shock is a severe, systemic hypersensitivity reaction to an allergen. Such allergic reactions can be caused by foods such as nuts, fish and dairy; drugs, including antibiotics and anaesthetics; and insect bites and stings.

COVID-19 and shock

Recent discussion has surrounded the clinical presentation of patients testing positive for SARS-CoV-2 and developing COVID-19 disease with hyperinflammatory states that share common characteristics with the signs of septic shock.

Patients with COVID-19 who have deteriorated have been observed to present with peripheral oedema, vasoplegic syndrome (decreased systemic vascular resistance) and disseminated coagulation leading to multi-organ dysfunction syndrome.

Critically ill patients are noted to be non-responsive to fluid resuscitation and to require further inotropic support to improve perfusion status (Dallan et al, 2020; Riphagen et al, 2020).

A case study described by Tavazzi et al (2020) involved the detection of COVID-19 in one patient presenting with COVID-19 symptoms such as respiratory distress and hypotension leading directly to cardiogenic shock; after initial stabilisation and treatment, the patient died later from diagnosed Gram-negative septic shock and organ failure.

While there is no doubt that further research and evidence is required to fully understand the effects COVID-19 has that bear similarities to the presentation of septic shock, a wealth of evidence has begun to appear that reports COVID-19 as the direct and root cause of a multisystem inflammatory syndrome in both adults and children, which requires similar treatment pathways.

However, the overriding pathogenesis and epidemiology of this newly discussed condition need to be studied further to understand its effects in both the short and long term (Morris et al, 2020).

Stages of shock

There are four stages of shock that occur sequentially as the patient's condition progresses and physiological changes begin to take place at a cellular level.

The first (initial), which has predominantly more acute clinical signs, and the second (compensatory) stages of shock are supported by the body's innate compensatory abilities to improve circulation and venous return to the heart. During the third (progressive) stage, symptoms worsen significantly as the compensatory mechanisms tire because of their increased workload. With further deterioration, the last (refractory) stage is reached, where the body fails to protect itself further, with organ failure, irreversible cell damage and the probability of death occurring.

Understanding these aspects of clinical decline and being able to identify each stage of shock as it develops will help nurses to prioritise and guide interventions with the objective of reducing both morbidity and mortality of patients (Urden et al, 2021; Tait, 2022).

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Initial

To maintain a relative state of homeostasis, the body employs the parasympathetic branch of the autonomic nervous system and uses acetylcholine to regulate involuntary physiological processes such as blood pressure, heart rate and respiration (Waxenbaum et al, 2021). These constant alterations occur through physical stimuli or small pathophysiological incidents, and are often so minimal that they are not consciously noticed.

Diagnosing shock at such an early stage is often difficult; the patient may be observed as feeling unwell and often appears pale and anxious (Hill and Mitchell, 2020). While the clinical signs are subtle, cellular change starts in response to a reduced cardiac output as cells begin to switch from aerobic to anaerobic metabolism.

Compensatory

As the original cause of shock develops (fluid loss, pump failure, ischaemia or widespread vasodilation), the body responds to the decrease in circulating volume and tissue perfusion through stimulation of the sympathetic nervous system, triggering a cascade of events in the form of hormonal, neural and chemically activated compensatory mechanisms designed to support blood pressure and readdress the imbalance between oxygen supply and demand. This is often referred to as the 'fight or flight' response to stress, trauma and states of inflammation where the body compensates in response to a threat, perceived or otherwise, to provide adequate blood flow to the vital organs such as the heart, kidneys, liver and brain to maintain homeostasis (Migliozzi, 2017).

As blood pressure falls because of poor cardiac output, sympathetic nervous system activity is initiated by the hypothalamus. This site within the brain is a key component in regulating blood pressure in response to both central and peripheral stimuli (Carmicheal and Wainford, 2015). The hormonal response to sympathetic activity is driven by the adrenal glands releasing catecholamines epinephrine and norepinephrine.

Epinephrine has two functions. First, it affects beta-1 receptor sites to improve both heart rate and the force of cardiac contraction to increase oxygen and nutrient delivery throughout the circulation. Second, it affects beta-2 receptor sites in the lungs to increase the respiratory rate and reduce airway resistance (Dutton and Finch, 2018) to improve oxygenation. The rise in both heart and respiratory rates are often noticeable, indicating that compensation has begun.

Norepinephrine affects the systemic and peripheral circulations by applying vasoconstriction. This resistance within the vasculature is intended to raise blood pressure by diverting larger volumes of blood towards the major organs to improve perfusion. Peripheral vasoconstriction is recognised by patients having cool and clammy extremities.

The initial low blood pressure is also recognised as a low blood flow throughout the renal system. The glomerular filtration rate falls if blood pressure is reduced to 60 mmHg within the glomerulus apparatus; this triggers a series of events known as the renin-angiotensin-aldosterone-system (RAAS) cascade. The intrinsic aim of this release of hormones (involving the kidneys, liver, lungs and pituitary gland) is to assist the kidneys to increase the reabsorption of water to increase blood volume, thereby raising blood pressure.

The juxtaglomerulus centre in the nephron releases the hormone renin, which stimulates the conversion of angiotensinogen, produced by the liver, into angiotensin 1. This is further metabolised into angiotensin 2 by angiotensin-converting enzyme (ACE) secreted from the lungs and the proximal convoluted tubules (Waugh and Grant, 2018).

The principle function of angiotensin 2 is to work as a powerful vasoconstrictor that helps to increase blood pressure. The presence of renin causes the release of aldosterone from the adrenal glands, increasing the reabsorption of sodium and water within the renal tubules to improve overall blood volume. This compensatory mechanism is augmented if fluid volumes rise inordinately within the heart chambers through fluid reabsorption, with atrial natriuretic peptide being released to inhibit aldosterone, thus creating an overall homeostatic balance between the promoted compensatory actions (Richards and Edwards, 2014; Waugh and Grant, 2018).

The neural pathway mechanisms for blood pressure control are predominantly found within the pons and medulla of the brain, with activation starting in response to internal cardiovascular changes such as vessel tone and cardiac output (Dutton and Elliot, 2021). A change in blood pressure is detected by the baroreceptors in the aortic and carotid arches; this triggers a neural response that causes norepinephrine to apply peripheral vasoconstriction.

Low blood volumes raise blood concentration and viscosity, which are detected by the osmoreceptors located in the hypothalamus. These promote the release of antidiuretic hormone, also known as vasopressin, from the pituitary gland, which increases the amount of water reabsorbed within the kidney tubules (Migliozzi, 2017), decreasing blood osmolality to improve blood flow. These responses are co-ordinated to raise cardiac output and blood pressure to support systemic organ perfusion.

A chemical response is activated by detection of changes to the blood pH by chemoreceptors, which are located peripherally within the aorta and carotid arteries and centrally within the medulla oblongata. These become activated when blood pH changes in response to alterations in circulating oxygen and carbon dioxide levels. A common sign of shock caused by a decrease in oxygen transport is the creation of an acidotic environment rich in carbon dioxide. The body's natural response to readdress this acid-base imbalance is to hyperventilate in an attempt to remove excess carbon dioxide waste and recruit more oxygen within the lungs. Alongside an increased respiratory effort, the heart rate is also stimulated to deliver what oxygen the body can maintain.

While the majority of shock types present with a tachycardia and tachypnoea, neurogenic shock is typically the exception to the rule and is associated with the opposite—bradycardia and bradypnea—because sympathetic nervous system pathways are interrupted so fail to trigger the compensation required (Dutton and Finch, 2018: Tait, 2022).

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Table 2. Shock type and NEWS2 observations

Shock type	A+B Respirations	A+B SpO_2 (Scale 1 and 2)	C Blood pressure	C Heart rate	D Consciousness	E Temperature
Hypovolaemic	Tachypnoea	Reduced	Hypotensive	Tachycardic	Alert	Cool
Cardiogenic	Tachypnoea	Reduced	Normal to hypotensive	Tachycardic	Confused/ responsive to pain	Cold
Obstructive	Tachypnoea	Reduced	Normal to hypotensive	Tachycardic	Altered/responsive to pain	Cool
Septic	Tachypnoea	Reduced	Hypotensive	Tachycardic	Confused	Warm
Neurogenic	Bradypnoea	Reduced	Hypotensive	Bradycardic	Altered	Warm
Anaphylactic	Tachypnoea	Reduced	Hypotensive	Tachycardic	Altered	Warm

NEWS2: National Early Warning Score
Source: adapted from Tait (2022)

Table 3. Targeted therapies for individual shock types

Type of shock	Therapy/intervention
Hypovolaemic	Treat underlying cause of hypovolaemia: Wound management/surgical intervention Endoscopy to assess gastrointestinal bleeds Intravenous fluid/blood products/tranexamic acid
Cardiogenic	Electrocardiogram: assess cardiac rhythm Echocardiogram: assess for cardiac filling and pump failure Percutaneous coronary intervention Intra-aortic balloon pump
Obstructive	Ultrasound/X-ray/echocardiogram: assess for the source cause Removal of pericardial fluid Needle decompression and chest tube Cardiac surgery
Distributive	Sepsis: treat infection—use Sepsis 6 or Surviving Sepsis Campaign guidelines Neurogenic: CT scan; treat underlying cause and provide pain relief Anaphylactic: treat effects of antigen with adrenaline, antihistamines, steroids; follow local anaphylaxis algorithms and guidelines

Sources: adapted from: Migliozzi 2017; Moruzzi and McLeod 2017; Silva et al, 2018; Hill and Mitchell, 2020; Jalota and Sayad, 2021; Stashko and Meer, 2021; Surviving Sepsis Campaign, 2021; UK Sepsis Trust, 2021; Resuscitation Council UK, 2022

Progressive

As the patient reaches a point of progressive shock, they are considered to be in a critical condition and usually require intensive organ support. If this is captured early, suitable and timely treatment can save life even at this juncture.

However, if the initial cause of shock is not addressed, the body's compensatory mechanisms will become overwhelmed, with cardiac output and blood pressure continuing to decrease. Tissue damage is likely to have occurred at this stage owing to continued hypoperfusion, with cell dysfunction creating a rise in lactic acid initiating metabolic acidosis.

Because of ongoing hypoxia, the cells switch from aerobic to anaerobic metabolism as they search for efficient energy stores to produce adenosine triphosphate to support their continued function. Inflammatory mediators histamine and bradykinin are also released, decreasing any preserved arterial vasoconstriction via their vasodilating nature, resulting in a further reduction in blood returning to the heart. Further hypoxic injury will result with depression of the vasometer centre, reducing sympathetic nervous system drive (Migliozzi, 2017).

If life-saving intervention is not initiated, the patient will deteriorate rapidly with the likely onset of cardiac failure, acute kidney injury, lung damage and poor cerebral perfusion, reducing their overall level of consciousness (Richards and Edwards, 2014; Dutton and Finch, 2018).

Refractory

This is also known as irreversible or end-stage shock. It often renders a patient unresponsive to treatment, however intensive, because of irreversible cellular damage and consequent multiple organ failure. At this stage, death is expected to be the most probable outcome (Urden et al, 2021).

Nursing assessment and interventions

Immediate clinical assessment of the patient is required to understand both the type of shock presented and to ascertain the stage of shock the patient is experiencing.

A standardised assessment of vital signs using the National Early Warning Score (NEWS2) (Royal College of Physicians, 2017) combined with the structured ABCDE (airway, breathing, circulation, disability and exposure) approach (Resuscitation Council UK, 2022) can determine a requirement for emergency intervention and provide critical information to help guide treatment and support care planning.

Thorough patient assessment also enables nursing teams to determine a baseline so future assessments can quantify improvement or deterioration. *Table 2* depicts a vital signs assessment as guided by the National Early Warning Score (NEWS2) observation chart; this assists health professionals to formally diagnose the specific type of shock as characteristic signs are often subtle and vary.

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Individualised therapies will be required to target and address each specific underlying cause of shock (*Table 3*). However, the immediate requirement universally will be to provide haemodynamic support to restore an adequate circulating volume to improve cardiac output to assist tissue perfusion and aerobic metabolism (Morruzzi and McLeod, 2017). As Waugh and Grant (2018) explain, perfusion of the major organs including the brain through either compensatory mechanisms or medical interventions is vital to help stabilise the patient's condition.

Oxygen

Oxygen therapy can be used in accordance with the British Thoracic Society (O'Driscoll et al, 2017) guidelines to improve oxygenation; however, oxygen therapy alone will not treat the underlying cause of hypoxaemia, which must be separately diagnosed and managed.

Oxygen, which can be prescribed to support blood oxygen levels, is recognised as a drug and therefore has to be administered to meet specific target saturation.

High carbon dioxide levels (hypercapnia) may occur because of hypoxaemia, hypoventilation or poor ventilation/perfusion mismatch or gas exchange related to certain comorbidities. Oxygen therapy can be administered to counteract this but is effective only with functional ventilation. Therefore, the diagnosis of either acute or chronic hypercapnic respiratory failure may require non-invasive ventilation or intubation and mechanical ventilation to support oxygen levels, alongside continuous monitoring of SpO_2 and arterial blood gas analysis to assess the effects of treatment (Dutton and Finch, 2018).

Arterial blood gas

A systemic condition such as shock will create changes in the respiratory and metabolic acid-base balance (Richards and Edwards, 2014).

Arterial blood gas interpretation can be used to determine both respiratory and metabolic status, helping to confirm and explain the reasoning for assessment findings and whether certain therapies (such as oxygen administration) are improving a patient's condition.

Rapid blood gas interpretation can provide useful information regarding gas exchange, pH levels, lactate and the ability or inability of the patient to maintain a regular acid-base balance (Moruzzi and McLeod, 2017).

Insertion of a designated arterial catheter allows continuous blood pressure monitoring to be performed as well as providing access for blood biochemistry sampling (Vincent and De Backer, 2013).

Fluids

If fluid resuscitation is deemed appropriate and the patient does not require immediate blood products, National Institute for Health and Care Excellence (NICE, 2017) guidelines suggest a rapid bolus administration of 500 ml intravenous crystalloid (containing sodium in a range of 130–154 mmol/l) over 15 minutes for the deteriorating patient. Such crystalloids include Plasma-Lyte 148, Hartmann's solution and sodium chloride 0.9%.

However, if shock is caused by fluid loss, it is important to understand the root cause of hypovolaemia to guide fluid resuscitation choice (Silva et al, 2018). If a major haemorrhage is confirmed, local protocols for emergency transfusions should be followed with fluid status being monitored closely using the relevant documentation.

Aseptic insertion of a urinary catheter can help to monitor output precisely, with an ideal output being 0.5–1 ml/hr, which is one indication that the patient is responding to treatment (Migliozzi, 2017).

A decision will need to be made on whether the patient should be kept nil by mouth if it is likely that surgery will be required.

Pharmacology

With patients experiencing shock, drug therapies will be administered primarily to improve cardiac function and output, boosting overall myocardial activity to increase organ perfusion.

Dobutamine and dopamine are inotropic cardiac stimulants used to improve the ability of the heart to contract and the force of contractions. Vasoconstricting drugs such as metaraminol, epinephrine and norepinephrine are intended to divert blood towards the major organs, supporting compensation which may have started to fail.

Ephedrine and antimuscarinic drugs such as atropine and glycopyrrolate all aim to increase heart rate, assisting with any decrease in the sympathetic response (Dutton and Finch, 2018; Joint Formulary Committee, 2022). If vasoactive therapy is required, a central venous catheter should be inserted, which can also be used to administer fluid therapies (Vincent and De Backer, 2013).

Conclusion

Shock is a complex clinical condition which can be life threatening; it requires urgent assessment, nursing intervention and a thorough understanding of the pathophysiological mechanisms (Vincent and De Backer, 2013).

The condition can arise because of numerous causes, and progresses when the body's homeostatic compensatory mechanisms fail. If not treated correctly, this can lead to a failure of the cardiovascular system, advancing to end-stage shock and death (Migliozzi, 2017).

It is therefore vital that nurses are able to recognise shock as it occurs and are take the correct steps to initiate a timely diagnosis and provide individualised treatment in accordance with the professional standards required by the Nursing and Midwifery Council (2018) *Code*.

This article serves to provide an introduction to the patient in shock with discussion regarding its aetiology, causes, types and stages to help nurses assess, recognise and manage patients with appropriate treatment and interventions, the overall aim being to improve patient outcomes and preserve life. **BJN**

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KEY POINTS

- Shock is a complex, life-threatening clinical condition that arises from acute circulatory failure
- Its pathological state is caused by the circulation being unable to deliver sufficient oxygen and nutrients to tissues and cells
- Shock is an umbrella term used to describe a medical emergency with four main causes: severe reduction in the circulatory volume; reduced cardiac output; obstruction to circulation; and altered distribution of blood flow.
- The four stages of shock are the initial, compensatory, progressive and refractory stages
- If shock is not treated in a timely, appropriate manner, death can occur
- It is vital that nurses can recognise shock and initiate interventions to treat it

CPD reflective questions

- How would you consider changing your practice and approach to ensure a thorough patient assessment of the signs and symptoms of shock is performed?
- What would you consider implementing in your place of work to help educate and ensure teams are aware of the signs of shock?
- Consider whether you able to locate both your local trust and national guidelines to steer a suitable nursing response for the patient experiencing shock

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